PENTRAXIN-3 AND ENDOTHELIAL DYSFUNCTION PARAMETERS IN PATIENTS WITH METABOLIC DYSFUNCTION-ASSOCIATED STEATOTIC LIVER DISEASE AND ARTERIAL HYPERTENSION

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In the structure of general morbidity metabolic-associated steatotic liver disease (MASLD) occupies one of the leading positions [1]. The presence of MASLD is believed to be associated with endothelial dysfunction (ED) development and cardiovascular disease (CVD) increased risk [2]. Oxidative stress is the key factor in the emergence of ED – the process of cell free radicals cumulation adversely affecting it's function and membrane integrity [3]. Currently the ED problem attracts many researchers, as it is one of the earliest predictors of the vascular wall morphological changes, not only at MASLD, but also at arterial hypertension (AH) as well. The ED presence in the peripheral, coronary, micro- and macrocirculation in patients with AH has been proven [4]. Recently, studies of the ED progression mechanisms in patients with comorbid course of MASLD and AH are relevant.

Objective – to explore the relationship between MASLD on the background of AH with pentraxin-3 (PTX-3) and ED parameters including endothelial nitric oxide (eNOS), endothelium-dependent vasodilation (EDVD), fibrinogen and uric acid (UA).

Materials and methods. We examined 102 patients with MASLD. They were divided into 3 groups: group 1 included 52 patients with isolated MASLD, group 2 – 23 patients with MASLD and AH stage I, and group 3 – 27 patients with MASLD and AH stage II. Control group (group 4) was formed of 20 apparently healthy people. The mean patients' age was 46.23 ± 9.3 years. All patients underwent a collection of complaints, anamnesis, physical and general clinical examination, measurement of blood pressure (BP), daily BP monitoring, PTX-3 levels and determination of

ED indicators (eNOS, UA, fibrinogen, EDVD). Patients with viral hepatitis, liver cirrhosis, alcoholic liver disease, and AH stage III were excluded.

Results. Analysis of the studied parameters revealed significantly higher levels of PTX-3 ($p = 0.001$), fibrinogen ($p = 0.01$), and UA ($p < 0.01$) and decreased levels of eNOS ($p < 0.05$) and EDVD ($p < 0.05$) in group 2 and group 3 in comparison with the group 1, as well as in comparison of these groups with the control group ($p 1< 0.01$, $p2 = 0.01$). Thus, the average eNOS levels in patients with comorbid pathology was (295.6 ± 35.67) pg/ml, (356.7 ± 28.34) pg/ml in the group of patients with isolated MASLD and (431.5 ± 25.65) pg/ml in the control group. The average PTX-3 levels in patients with comorbid pathology was (421.9 ± 31.4) pg/ml, (254.3 ± 44.4) pg/ml in the group of patients with isolated MASLD and (53.2 ± 14.3) pg/ml in the control group. The average of EDVD levels among examined patients were the following: (2.11 ± 0.67) % in patients with MASLD and AH, (2.15 ± 0.48) % in the group of isolated MASLD, and (3.07 ± 0.56) % in the control group. The following average UA level were defined: (406.4 ± 25.6) μmol/l in patients with comorbid pathology, (369.3 ± 27.8) μmol/l in the group of patients with MASLD and (308, 9 ± 15.7) μmol/l in the control group. The average level of fibrinogen in patients with the comorbid course of MASLD and AH was (4.4 ± 1.4) g/l, (3.8 ± 1.3) g/l in patients with isolated MASLD and (2, 4 ± 1.1) g/l in the control group. In addition, significantly decreased levels of eNOS ($p = 0.01$), EDV BA ($p = 0.01$), and increased levels of UA ($p < 0.01$) and PTX-3 ($p < 0.01$) were found in patients with MASLD against the background of AH stage II in comparison with patients with MASLD and AH stage I. However, the relationship between the fibrinogen ($p > 0.05$) levels and the progression of the AH stages have not been confirmed in our study.

Conclusions. Our findings indicate the direct relationship between the PTX-3 and ED parameters involvement in ED developing and the further progression of MASLD. Also the obtained data indicate the relationship of AH and its stages with the ED development in patients with MASLD and AH comorbid course.

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